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Treatment of Hyperesthetic Neuropathic Pain in Diabetics

Decompression of the Tarsal Tunnel

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Objective
The authors evaluated the causal relationship between entrapment of the posterior tibial nerve and neuropathic pain and describe the results of nerve decompression in a selected group of patients with intractable pain.

Summary Background Data
Painful metabolic neuropathy has, until recently, been thought to be an irreversible and essentially untreatable complication of diabetes. Recent studies have shown that metabolic deterioration is only one component of the disease process.

Methods
A group of patients with intractable painful neuropathy and a positive percussion sign underwent posterior tibial nerve decompression.

Results
Nerve decompression relieved the pain in the majority of treated patients. Return of other sensory function also was noted.

Conclusions
Painful diabetic neuropathy of the lower extremities is potentially reversible. It appears to be caused partially by nerve entrapment and can be reversed by decompression.

Painful neuropathy in diabetes has been the subject of extensive clinical and basic research.[1–3] Until recently, neuropathic degeneration was thought to be entirely caused by neuronal degeneration related to chronic metabolic abnormalities.[4,5] However, this did not explain the occasional serendipitous disappearance of pain after an operation on the foot and ankle in these patients. Recently, it has been suggested that compression of nerves may play a role in the neurologic deterioration associated with the loss of sensory-motor function in patients with long-standing diabetes mellitus.[6–8]

The purpose of this study was twofold—first, to evaluate a possible causal relationship between entrapment of the posterior tibial nerve and neuropathic pain that develops in patients with diabetes mellitus; second, to de-

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scribe the results of the treatment of a selected group of diabetic patients suffering from intractable pain due to neuropathy using operative tarsal tunnel decompression to relieve the symptoms.

METHODS

Twenty-six patients with painful neuropathy and diabetes mellitus were selected from a diabetic clinic devoted to foot care. There were 11 men and 15 women. The mean age was 59.6 years (range 43–73 years). All were adult-onset diabetics who had been treated for their disease for a mean of 16.2 ± 8.8 years (range 2.5–32 years). A positive percussion sign (Tinel's) was demonstrated preoperatively in 32 of 33 extremities . Eighteen patients required insulin to control diabetes. Six were being treated with oral hypoglycemic agents, and two patients' diabetes were controlled with diet only.

The patients were considered candidates for tarsal tunnel decompression based on the following criteria: presence of diabetes, absence of signs of lower extremity ischemia tissue (indicated by the presence of posterior tibial and dorsalis pedis pulses), absence of signs of infection, and the presence of persistent neuropathic pain for a minimum of 6 months, which, by history, clearly interfered with the patients' life style. The presence of a positive percussion sign (Tinel's) at the level of the tarsal tunnel also was used. The occurrence of neuropathic ulcers were noted both before and after treatment. The patients were further screened using standard electrodiagnostic studies, tests of two-point discrimination and measurements of pressure on plantar surfaces of the affected extremity. Tests of two-point discrimination were performed using a Disk-Scriminator (Baltimore, MD). Two point discrimination was recorded in millimeters. Electrodiagnostic studies were carried out in a standard fashion, but with emphasis on any likely nerve compression components. Plantar pressure measurements were completed using the EMED-SF system and novel*gmbh* software (Novel, GmbH, Munich, Germany). The measuring system consisted of a matrix of capacitance-type transducers platform, with 2736 sensors (4/cm^2) and a sampling rate of 50 Hz. The output from the EMED-SF platform was processed by the EMED pedobarograph microcomputer and the data were stored on a 3.5″ diskette. The data were subsequently transferred to a Compaq Pro 486 personal computer hard drive and analyzed using the EMED Multimask software (GmbH).

Pain was assessed using a 10-point scale ranging from 1 to 10, with 1 being barely perceptible and 10 being intolerable. Qualitative assessment of the pain was obtained by questioning the patients about their perceptions with pain characterized as being aching, burning, shock-like, continuous, or intermittent.

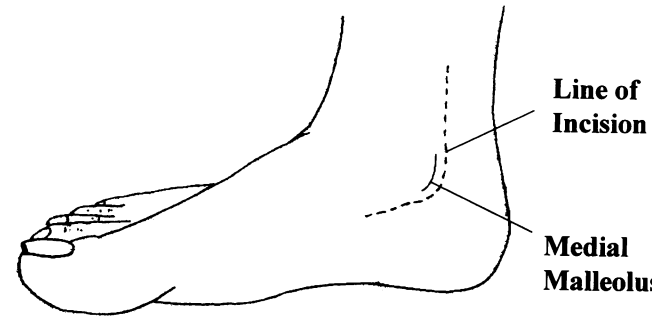

Figure 1. The skin incision extended from approximately 5 cm above and 1 cm posterior to the medial malleolus to a point 1 to 2 cm below and 2 cm anterior to the malleolus.

After informed consent was obtained, the patients underwent operative decompression of the tarsal tunnel using the following technique, modified from Pfeiffer.[9] Prophylactic perioperative antibiotics were administered, and the patient was placed in a supine position on an operating table. Tourniquets were not applied. After preparation of the skin, 20 to 30 mL of 1% lidocaine without epinephrine was injected subcutaneously along the path of the posterior tibial nerve above and below the medial malleolus. The skin incision extended from approximately 5 cm above and 1 cm posterior to the medial malleolus to a point 1 to 2 cm below and 2 cm anterior to the malleolus (Fig. 1). Sharp dissection was used to define the retinaculum (Fig. 2). An incision was made in the retinaculum and extended proximally the entire length of the structure. Distally, the retinaculum was incised to the point where the medial and lateral plantar nerves divided and followed a course deep into the plantar surface of the foot. All nerves were isolated and freed from any constraining tissues and vessels that could possibly cause compression (Fig. 3). Internal neurolysis was not performed. Significant varicosities were ligated. The calcaneal branch of the nerve was identified and traced distally for a minimum of 1 to 2 cm. Once all segments of the tibial nerve passing through the tarsal tunnel were decompressed, careful hemostasis was obtained. Then, only the skin was closed. The patients were kept at general bed rest overnight; however, unless significant swelling developed, progressive ambulation was permitted on subsequent days. Sutures were removed 3 weeks postoperatively. Postprocedure assessment consisted of electrodiagnostic studies, two-point discrimination, EMED evaluation, assessment of pain relief, and notation of any new ulcer formation.

Postoperative follow-up has averaged 13.4 ± 7.5 months (range 2–26 months).

RESULTS

Twenty patients were recorded as having pain relief at the time of the operation. Twenty-two patients had relief

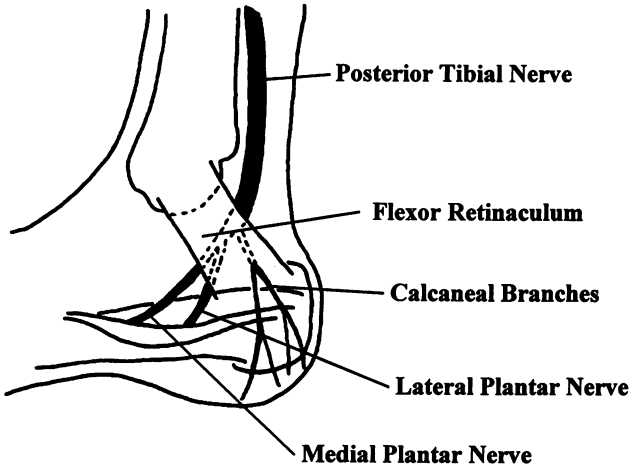

Figure 2. Sharp dissection was used to find the retinaculum.

of neuropathic pain within 7 days after their operation, and pain subsided in 24 of the 26 patients within 1 month (92%). Patients who had bilateral procedures had essentially identical responses on both sides. A positive percussion sign was elicited in all but one extremity.

The patients were questioned during the procedure. The completion of the operation was related to the achievement of pain relief. If, after the initial decompression of the posterior tibial, calcaneal, medial, and lateral plantar nerves, the patient did not describe relief, the procedure was extended proximally and distally to improve the decompression. In several cases, this produced benefit. In patients who had pain relief, no recurrence of burning pain has been reported to date.

No significant information was obtained from electrodiagnostic testing. All patients showed mixed axonal abnormalities with evidence of demyelination. No consistent relationship was found between localized symptoms and nerve distribution. Preoperative and postoperative nerve conduction test results did not vary markedly in any patient.

The EMED results also were not easily interpretable, primarily because preoperatively, the patients had severe pain and were unable to cooperate with the standard test format. Postoperatively, the patients were able to remain in contact with the mat for a longer time, but were not good candidates for evaluation because of their generally inconsistent gait patterns.

Two-point discrimination measured preoperatively and 1 to 6 months postoperatively indicated a pattern of overall improvement in tactile sensation in 19 of 26 patients (72%). Two-point discrimination preoperatively averaged 13.7 ± 2.9 mm for the group and 15.1 ± 4.0 mm for the respondents. Postoperatively, the mean was 11.7 ± 2.5 mm and the respondent was 11.1 ± 3.5. The average improvement was 4.0 mm in the respondent. Three patients'conditions worsened postoperatively. These patients continue to be tested periodically to determine long-term benefit. Nine patients reported subjective re-establishment of tactile sensation in their feet and toes during the operative procedure. In certain cases, this was quite dramatic. Pain relief was reported to occur simultaneously with improved tactile sensitivity, indicating that the pain relief was unlikely a result of local anesthetic injection. Thirteen patients had neuropathic ulcers at the time they underwent nerve decompression. Only one patient who did not have a foot ulcer preoperatively developed an ulcer on a treated extremity within the study period.

In all patients, preoperative pain was described as burning and averaged 8.9 (range 6–10) on the severity scale. Postoperatively, two patients (8%) had no relief. Those patients in whom pain recurred did not classify their discomfort as burning pain. Those patients (19.2%) noted aching pain of a minor nature (1 on a scale of 10). It is not clear from the study whether the patients could not distinguish the presence of two different types of pain preoperatively or whether improved sensation after nerve decompression allowed them to sense previously unrecognized noxious stimuli.

Operative findings that could have contributed to nerve compression were noted. These included inflamed tissues around the nerve, fibrosis, crossing vessels, varicosities, a tortuous or kinked nerve, or direct compression of the nerve by the retinaculum. Most patients had a combination of these operative findings, with inflammation and fibrosis predominant.

COMPLICATIONS

Four patients developed superficial wound infections that led to delayed healing. All wounds ultimately closed.

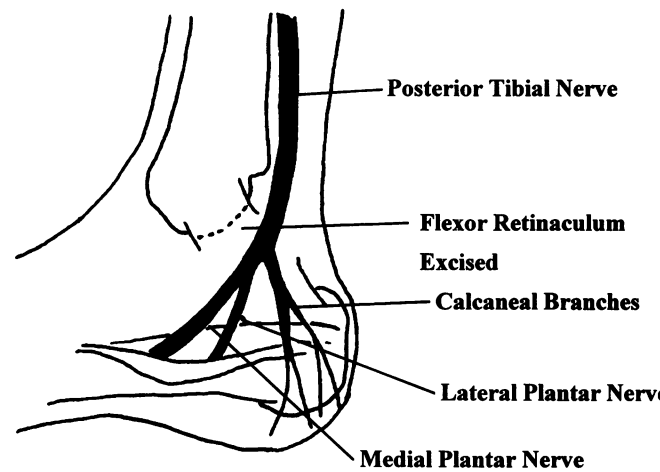

Figure 3. All nerves were isolated and freed from any constraining tissues and vessels that could possibly cause compression.

One patient developed numbness in the left fifth toe after a repeat decompression. The second operation was performed because the patient had underdone a successful procedure, only to have the pain reoccur after her ankle had been run over by an automobile. Complete pain relief was achieved after a repeat procedure, but the fifth toe did not regain sensation. It is not certain whether this was because of the auto accident or was iatrogenic.

The only patient who underwent decompression despite the absence of pulses developed a wound infection which healed. Unfortunately, the patient's overall condition deteriorated, and she ultimately required dialysis and treatment for heart failure. The patient developed lateral foot ischemia approximately 3 to 4 months after decompression and underwent amputation of the extremity 6 months postoperatively. We do not believe that there was approximate relationship between the patient's operation and the loss of the limb or other subsequent diabetic complications.

DISCUSSION

The results of this study demonstrate that relief can be obtained from the incapacitating dysesthesia associated with diabetic neuropathy. The results are consistent with the hypothesis—developed by Dellon—that diabetic neuropathy has multiple causal components and that compressive neuropathy may be the most significant component in the evolution of symptomatic neuropathic complications.[10]

The present series indicates that prompt and sustained relief from neuropathic pain can be achieved using nerve decompression in a selected subset of neuropathic patients who have evidence of viable but irritable nerves.

Although it is possible that other factors could account for the pain relief described, several indirect findings suggest that the decompression itself was responsible for the responses noted. The promptness of the pain improvement after decompression of the nerve is similar to that seen in carpal tunnel syndrome. The fact that no clear signs of extremity ischemia were present would mitigate against major vascular compression in the tarsal tunnel as the cause. The fact that other sensation, deep pain, and two-point discrimination were improved implies that other neurologic activity was improved by the procedure. The fact that pain relief has been sustained would make the natural tendency of these symptoms to wax and wane—an unlikely explanation for the observed results. This is quite different from standard teaching, which suggests that once neuropathy develops, it essentially is irreversible and no effective treatment exists. There is no question that a significant metabolic abnormality exists in the nerves of these patients. Sorbitol accumulation leads to decreased electrical function and also is at least partially responsible for the nerve and tissue edema that produces the compression syndrome. On reflection, it seems quite reasonable that the burning neuropathic pain is related to local nerve compression and ischemia. The symptoms described essentially are identical to those that commonly occur when temporary nerve compression occurs in nondiabetics. The difference between these acute episodes and the chronic symptoms in diabetes relates only to the persistence of the local ischemia caused by compression. Over an extended period of time, continued ischemia leads to secondary phenomena, including inflammation, scar formation, and myelination, which intensify symptoms and ultimately result in permanent destruction of the nerve.

Patient selection is critical in successfully using tarsal tunnel decompression to obtain pain relief. Dellon showed that identification of a viable nerve using a percussion sign (Tinel's) was an excellent predictor of potential success. In his study, he reports an 80% chance of improvement if a positive percussion sign is present and only a 50% chance if not. One of our two failures was in a patient who had been in pain for more than 5 years and who had a negative Tinel's sign. This is not to say that improvement cannot be obtained in patients without demonstrable nerve irritability, but the likelihood of success appears to be diminished and there is a risk to the extremity that must be considered when performing an operation. A steeply declining risk/reward ratio should indicate the need for caution in the application of this technique until persistent severe symptoms warrant such intervention.

This study confirms that standard electrodiagnostic studies are not helpful in defining patients with compressive neuropathy who may be candidates for operation. Similarly, plantar pressure measurements do not appear to have significant predictive value. Improved two-point discrimination was noted after tarsal tunnel decompression. The overall magnitude of improvement measured during our study period was not great. However, restoration of functional neurologic status may require an extended period of time. Long-term assessment of these patients will be required to determine the extent to which two-point discrimination can be restored by nerve decompression. Although no method of selecting the responding group was identified, the fact that it was possible to quantitate the improvement provides a basis for hope that "protective sensation" can potentially be restored.

Only one patient in this series has manifested a new ulcer after successful decompression. Long-term studies will be needed to confirm the value of the procedure as a method of ulcer prevention. If it is possible to diminish the risk of ulcer formation, tarsal tunnel decompression

will have a much more significant role in the treatment of diabetic foot disease than pain control alone.

This study supports the seminal work of Dellon regarding the multicausality of diabetic neuropathy. It also suggests that successful treatment of painful neuropathy is possible on a consistent and sustained basis in a properly selected group of patients. The fact that multiple wound infections occurred in the series should reinforce the need for careful patient selection. Only people with prolonged incapacitating pain should be considered. The full extent of the value of these concepts and treatment, with respect to limb preservation, remains to be determined.

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Discussion

DR. LEWIS M. FLINT, JR. (New Orleans, Louisiana): Thank you, Dr. Jurkiewicz. I appreciate the opportunity to review the manuscript.

I would venture a guess that of the diabetic patients we discharge from our inpatient services, those patients having complications of neuropathy of the feet don't get discharged walking better than they did on admission, by and large. Most leave having had an amputation or other incomplete treatment of their problems. Through dedication and interest,

Dr. Wieman has developed a service which strives to keep diabetics walking. The data that he has presented today are testimonies to his success. I have four questions for the authors:

Number one: Is outcome related to the control of blood sugar levels in this patient group?

Second, do you have histologic or histochemical examinations of nerves which did not respond to decompression which might disclose important differences which would be related to outcome?

Third: Are your patients receiving any specific medical therapy for neuropathy as distinct from their diabetes? And does this figure in with outcome and/or postoperative management?

And, finally: Does decompression change other symptoms of neuropathy such as loss of position sense?

Thank you very much.

DR. LUIS O. VASCONEZ (Birmingham, Alabama): Dr. Jurkiewicz, Dr. Copeland, distinguished Members and Guests of the Association.

Drs. Wieman and Dr. Patel bring to our attention a most difficult clinical problem. Diabetic neuropathy, when it presents in the feet, is followed by trophic ulcerations in the weight-bearing surface with advancing infections, which are most difficult to treat. A good number of patients, as demonstrated by the authors, may have severe pain and discomfort. The decompression of the tarsal tunnel in these patients is a very attractive idea, particularly if one equates or compares it with the decompression of the median nerve at the wrist. Unfortunately, the improvement and results are not the same. The authors' data to me demonstrate that the tarsal tunnel decompression is effective in a select number of patients, but the indications for choosing the proper patients who will benefit from the procedure is not quite clear to me. I question a number of statements by Dr. Wyman and they include the following:

I do not think that the use of palpable pulses in diabetics is an accurate measurement of adequacy of vascular supply. Diabetic patients often have calcified vessels. A better measure would be ankle to brachial indices. This is important if one is to hope that the wound will heal primarily.

The authors indicate that the neuropathic pain often was relieved on the table. Yet, they used local anesthetic before making the incision in relatively large amounts (20 to 30 cc). One could conjecture that some of this anesthetic will diffuse around the nerve. Similarly, for an adequate decompression, often one has to free up the origin of the abductor pollicis muscle, and this could be quite painful. Although the authors decompress only the tarsal tunnel, in some patients who also had pain in the dorsum of the foot, pain in the entire foot was noticed to disappear after the decompression. How is that possible?

Their data on two-point discrimination suffers because there are no statistical studies to see if, indeed, there is a difference.

It is important to know if the patients with preoperative ulcers subsequently healed, which would be a demonstration of some return of protective sensibility.

A number of the wounds did not heal—4 out of 26 patients—which is relatively low considering the type of patients. Importantly, there was no relief of symptoms in two patients, and five additional patients (27%) had recurrence of pain in the follow-up period. Therefore, conclusions are tenuous, particularly because the follow-up period is relatively short (13.4